Letters to the Editor


Presence of the epidemic European fusidic acid-resistant impetigo clone (EEFIC) of Staphylococcus aureus in France—joint authors’ response


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Sir,
The finding by Laurent et al.1 of isolates belonging to the epidemic European fusidic acid-resistant impetigo clone (EEFIC) in France is interesting as this observation is somewhat in contrast to our findings in the EPISA study.2,3 However, as proposed in the EPISA study by Denton et al.,2 the different proportions of patients suffering from impetigo in the UK (120/461; 26%), Ireland (49/449; 11%) and France (36/480; 7.5%) could be associated with ‘cultural differences in patients seeking medical assistance’. Laurent et al.1 also point to this explanation as the French people may prefer to consult specialists or hospitals if affected by impetigo. In the EPISA study, 25 Staphylococcus aureus isolates obtained from French impetigo patients were characterized, but in contrast to the UK and Ireland, none of these isolates belonged to the EEFIC.2,3 Thus, although impetigo cases may be more numerous in France than found in our study, the dissemination of the EEFIC may still be limited. The latter is confirmed by the results of Laurent et al.,1 since they only found 10 EEFIC isolates (extrapolated: 25–50 isolates) after thorough molecular investigations on a large collection of S. aureus spanning an 8 year period and originating from hospital as well as non-hospital (community) laboratories.

Influenza virus infection: don’t forget the role of the mucociliary system!

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Sir,
In a recent issue, Zhang1 summarized the interest in animal models to study the relationship between the influenza virus and secondary pneumonias. We agree with the role of a preceding influenza infection in secondary pneumonia.2–4 Referring to studies by McCullers’s group,5–7 Zhang speculated on the adherence of Streptococcus pneumoniae in the lungs after cleavage of sialic acid residues from the surface of host cells, exposing cryptic receptors to S. pneumoniae and allowing bacteria to adhere. However, he did not mention that McCullers stated in 2006, ‘the receptors that pneumococcus utilizes to adhere and invade in the lung are currently unknown’.8 As these receptors have been searched for, but not found, it is tempting to suggest that perhaps they do not exist. Thus, they cannot be a reason to predict risks in the case of the therapeutic use of a sialidase fusion protein.

In the same issue, Nicholls et al.9 supported the therapeutic use of sialidase fusion proteins. These authors made a clear distinction between the secondary S. pneumoniae infection in

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References


relation to a viral neuraminidase and the secondary effects of influenza virus infection in relation to airway epithelial damage, considering that, from a scientific point of view, only the latter was significant. In our opinion, this distinction is not appropriate because the disorders produced by viral neuraminidase are important components of the epithelial damage to the airway.

Both points of view are interesting but obviously incomplete:

- the physiological role of sialic acid receptors is to allow adherence of the mucus that protects the epithelial tissues from dehydration, microbial pathogens and reactive oxygen species produced by infectious bacteria and/or the oxidative burst of leukocytes;
- the mucus line of defence comprises a viscoelastic gel that immobilizes bacteria and virus, which are then cleared by the ciliary movements;
- mucoproteins are either secreted or membrane-tethered, and sialoglycoproteins mediate the cell adherence and the visco-elasticity of mucus, and serve as receptor sites for the binding of exogenous macromolecules;
- viral sialidase (neuraminidase) seems to facilitate the spread of the virus by limiting their attachment to the cells and to mucus layers;
- in the early stages of influenza infection, respiratory cells are modified: ciliary function decreases, viral neuraminidase is expressed at the surface of epithelial cells and the neuraminidase lowers the adhesion of mucus, leaving the cells unprotected and allowing bacteria to develop at their surface;
- statistically, in these circumstances, the first bacterium to invade airways is Haemophilus influenzae, probably as a consequence of adhesins expressed in non-typeable strains of H. influenzae, and as indicated by Pfeiffer’s belief that it was the causative agent of influenza. However, other bacteria are very often encountered. These include S. pneumoniae, which was extensively studied because it was capsulated and more pathogenic. In conclusion, we feel troubled by the fact that the authors neglected the role of the mucociliary system as an important line of defence against influenza viruses, because it seems to be the major link to secondary infections (pneumonia, sinusitis or otitis media), and we suggest that the effect of a sialidase fusion protein treatment on the mucous membrane and on the clearance of infectious bacteria should be studied.

Transparency declarations

None to declare.

References


2. Van der Slujs KF, van Elden LJ, Nijhuis M et al. IL-10 is an important mediator of the enhanced susceptibility to pneumococcal pneumonia after influenza infection. J Immunol 2004; 172: 7603–9.


